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SOREMOUTH (CONTAGIOUS ECTHYMA) IN SHEEP AND GOATS

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**In cooperation with U. S. Department of Agriculture.

Soremouth (Contagious Ecthyma) in sheep and goats is an infectious disease and occurs especially in young animals. It is quite prevalent on the range in the sheep and goat-raising area in western Texas and elsewhere. Sometimes it is also very troublesome among feeder lambs in the feed lot. Older animals may also be infected artificially.

The disease manifests itself by a swelling of the lips followed by papules, vesicles, pustules, and scab formation so that the lips in the end become stiff, unpliable, and covered with crusts. The crusts are finally shed and the lesions heal without leaving a scar. The disease itself is not very fatal but losses from screw-worm infestation of the lesions, from reduced thriftiness of the young animals, or from shrinkage in the feed lots may be very heavy.

The virus of the disease is not known. It has been reported as filterable. Our results confirm those of others that filtration is difficult to obtain. The lesions remain localized irrespective of the site of inoculation. Our results and those of others indicate that one attack of the disease leaves a well marked immunity and that such an immunity develops irrespective of the location of the lesions on the skin. We propose to utilize this fact and inoculate the young animal on some part of the body other than the lips before screw-worm time arrives.

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SOREMOUTH (CONTAGIOUS ECTHYMA) IN SHEEP AND GOATS*

H. Schmidt and W. T. Hardy

In the sheep-raising area in the western part of the State a condition occurs among sheep and goats that the ranchmen refer to as soremouth. This condition, manifesting itself mainly in a painful swelling of the lips and the appearance thereon of papules, vesicles, and pustules which soon break, after which the affected parts become covered with thick, tightly-adhering grayish-brown crusts, has been known to the ranchmen for a number of years but until recently efforts had not been made to study the disease, mainly because the mortality, as a rule, had remained very low. Other losses, however, resulting from this trouble such as numerous cases of screw-worm infestation, losses due to undernourishment in affected young lambs and kids, and heavy shrinkage in the feed lot, if the disease should break out there, have brought forth requests by ranchmen that something be undertaken to avoid these occurrences. Since, in our opinion, the cause and nature of the disease were still in doubt, experiments were planned and started at the Ranch Experiment Station early in 1928 and later participated in by the Main Station at College Station with the view of studying the nature of the disease and to find some practical remedy for it.

In the older literature a similar disease has been described as necrobacillosis (10, 11), lip-and-leg ulceration (14), etc., and attributed to the *Actinomyces necrophorus*. There is no doubt that this organism has been found in the lesions of the disease described but it is not clear whether it has been established as the primary cause or whether it was only present as an occasional secondary invader. The photographic illustrations accompanying these discussions showed lesions of the mouth of sheep and goats strikingly similar to those observed by us, but other lesions, notably ulcers on the legs as reported by Mohler, were not observed by us.

This was the extent of the literature available to us at the time our first experiments were undertaken. As current literature became available it was searched and a number of references found that threw additional light on the subject. Jacotot (1), in 1924, speaks of a contagious ecthyma of the lips of goats in Annam, Indo-China, and describes lesions that resemble those observed by us. He pointed out that the submaxillary lymph glands are severely inflamed and that recovery leaves a durable immunity. In 1928 Theiler (2) published his observations on contagious ecthyma of sheep and goats as observed in South Africa and also reviewed some literature on the subject. The lesions as observed by Theiler also closely parallel those observed by us. Theiler could verify the findings of others that the disease could readily be transmitted from animal to animal by lightly scarifying the skin and applying some of the emulsi-

*Part of the work carried on by E. A. Tunnicliff, resigned December, 1929.

fied material from the lesion, an observation which in the meantime had also been made by us. He records that Zeller, Blanc, and Aynaud,* succeeded in filtering the virus, although Zeller did not succeed in all cases when using Berkefeld filters V or Nordtmeyer Berkefeld filters. Aynaud used Chamberland filters L1 and L2 bis, and Blanc used Chamberland L2 filters. Theiler himself obtained negative results in his filtration experiments when using Seitz asbestos filters. In a later publication (15) Jacotot reports that he succeeded in filtering the virus of contagious ecthyma of the goat through Chamberland filters F and L2. In 1928 Melanidi and Stylianopoulo (13) published on contagious ecthyma of sheep in Greece and reported that this disease had not hitherto been observed in that country (Blank and co-workers reported it from Greece in 1922) and expressed the belief that the disease was introduced with importations of sheep from Syria and northern Africa. In this outbreak the losses varied from 5% to 60% and were mostly due to inanition and pulmonary or intestinal complications. The authors point out that the disease resembled that described from France in every respect; that it was characterized by the formation of ulcers and scabs on the lips, nostrils, and buccal cavity; that these eruptions were preceded by a fever; and that the regional lymph glands were affected. These authors speak of vaccinating against this disease with good results.

Glover (12) in 1928, reported the results of some of his work with contagious pustular dermatitis or stomatitis (Contagious Ecthyma) of the sheep in England and gave a description of the microscopical skin lesions. He found that the virus is filterable with difficulty and that sheep and goats alone are susceptible to it; that it is highly resistant to desiccation and that animals recovered from an attack of the disease remain resistant to further attacks for at least eight months. He could transmit the disease with a few drops of suspension made with dry scab material diluted 1 to 50,000. He could not transmit the disease by intradermal inoculation of blood drawn after pustules had formed. Neither could he produce skin lesions of the disease when he inoculated the virus subcutaneously or intravenously. His attempts to transmit the disease to the rabbit, guinea pig, mouse, fowl and pigeon were all negative. Glover also reports, that in the experience of Aynaud, if sheep are inoculated on the thigh with suitable material which produces a benign local reaction there need be no apprehension of a generalisation of the disease.

Carre (8), in a discussion of some diseases among sheep, states that Aynaud established that ecthyma and ulcerous stomatitis were manifestations of the same disease, which was caused by a filterable virus. According to this author ulcers may develop about the mouth, gums, pharynx, and oesophagus and even inflammatory patches in the intestines and involvement of the lungs may be observed. Such lesions were more recently reported by Newsom and Cross (6) as complications of soremouth in lambs. According to these authors "there is still some question about the etiology of soremouth but there can seemingly be no doubt that these

*The publications of these three authors are not available to us.

complications are due to invasion of *Actinomyces necrophorus*." According to Carre (8), opinion is divided as to an immunity. The disease naturally acquired is said to give immunity for 2½ years, while sheep experimentally infected acquire the affection after one year.

Ulcers and similar lesions on the digestive mucosa of sheep and goats had already been reported by Miegeville (9) in 1927, from Morocco. This author states that the disease is known as contagious ecthyma of the lip or ulcerous stomatitis, but is of opinion that two distinct diseases are involved, namely, contagious ecthyma occurring especially in adults and ulcerous stomatitis occuring especially in lambs and kids, which latter form may involve the mucosa of the mouth, pharynx, oesophagus, and intestines, and also the lungs. Willems (3), in 1929, speaks of contagious ecthyma of the lips of sheep as occurring in Belgium and suggests the possibility of its transmission to man. In 1930 Hatziolos (4) reported his observations on an epizootic of contagious ecthyma of the lips which he observed during the warm season in 1929 occurring among sheep over a large part of Greece and in which he reports losses ranging from 5% to 60%. In his observations the disease remained confined to sheep and was not transmitted to goats living in contact with affected sheep. He, too, reports the transmission of the affection to man and observed the occurrence of stomatitis with vesicles on the gums, tongue, cheeks, and lips of the shepherds and their children. He also speaks of vaccinating the sheep much in the same way as it is practiced in the human subject against smallpox and gives a simple method of preparing the vaccine. Vaccinated animals left in contact with sick ones did not contract the disease.

In 1930 Seddon (5) describes the disease from Australia. This investigator was able to reproduce the disease with filtered material and states that the virus is too small to be seen with the highest powers of the microscope. He also reports that lesions sometimes occur on the inside of the lips, on the gums, dental pad, or palate. He observed that sheep which have recovered from an attack of the disease are immune from further infection and that sheep may be artificially immunized by vaccinating them much in the same way as the vaccination is carried out for the human subject against smallpox. Recently Howarth (7) reported an outbreak of the disease from California. He could readily transmit the disease to sheep and a goat by applying emulsified scabs or other infective material to the scarified skin. He was unable to find any evidence of the presence of *Actinomyces necrophorus* either in cultures or animal inoculation experiments. On the other hand he succeeded in passing the virus through a Mandler and Chamberland L2 filter but speaks of the difficulty encountered in obtaining uniform results. Attempts to infect three guinea pigs, two rabbits, two pigs, a dog, and a cow gave negative results in all cases.

THE DISEASE AS IT OCCURS NATURALLY

In our investigations we have found that soremouth in the field usually occurs only in young animals, either in the suckling lamb or kid or after the lamb or kid has been weaned or has probably been placed in the feed lot. Only rarely are cases observed in older animals. The first symptoms that the ranchman usually observes are swelling of the lips, which soon become more or less inflamed and are later covered by more or less profuse scab formation. A closer examination will show that the swelling when it first appears is usually confined to the lips and that the skin of these also shows the formation of papules, vesicles, and pustules. These lesions need not remain confined to the lips, but may spread either to the buccal mucous membrane or to the hairy skin farther removed from the lips. In the latter case they, nevertheless, remain confined to the nose or the cheeks. These scabs or crusts will become thicker as time goes on and may reach a thickness of one-half inch or more. They are dry and grayish-brown to dark-brown in color. Their surface may become deeply-fissured and cracked. These cracks are sometimes so deep that the whole mass of crusts may appear as two or more warty protubances. In the course of three or four weeks such scabs will gradually crumble away and finally disappear, the condition healing without leaving a scar. If, however, such scabs are forcibly removed either accidentally by the animal itself or otherwise, a painful, raw, bleeding surface will be left, which in the course of time will become covered with new scabs which, in due time, will drop off, and the lesion will have healed without leaving a scar.

The lesions do not always show such a grave character as just described but rather vary a great deal from season to season, from flock to flock or even within the same flock. Often we see only insignificant scab formation at varying places on the lips, but perhaps most frequently at the commissures.

We have observed the development of lesions on the udder, under the tail, and on the ears. Other observers have reported them on the udder, on the under surface of the tail, on the feet, on the thighs, and in the axillae. From these observations it appears that unless the lesions appear on the lips or feet they are frequently found on that part of the skin devoid of wool or hair.

The inflammation of, and the marked scab formation on, the lips leaves these in a stiff, leathery, unpliable condition, every movement of which is more or less painful, thus greatly interfering with the suckling of the young or with the prehension of food to the extent that affected animals lose in condition and sometimes even die. Moreover, since soremouth occurs in the season of the year when the screw-worm fly is most active, screw-worm infestation of the affected parts is rather common. Indeed, the losses from screw-worm infestation, both in labor involved in caring for such cases and loss of animals as the result of such screw-worm infestation, is perhaps greater than that caused by the soremouth condition itself.

THE DEVELOPMENT OF THE DISEASE FOLLOWING INOCULATION

The development of the lesions can best be studied on the soft skin devoid of wool or hair as it is found on the under surface of the tail or on the internal aspect of the thigh in the sheep or the goat. If this area be carefully and thoroughly cleaned, then lightly scarified so as to break the superficial epidermis but not deep enough to draw blood and then an emulsion of virulent virus be applied, the development of the different stages of the lesions can be clearly observed. On an area thus inoculated one will see within the first 24 to 48 hours a slight reddening and swelling of the skin along the line of scarification, which may increase in intensity during the next day or two. Upon careful observation one may at this time also see along the line of scarification, small circumscribed areas, which are slightly raised above the general level of the skin. These are called papules and represent a marked, sharply circumscribed infiltration of the skin. At the tip of each papule a small vesicle soon appears which at first is filled with a clear, transparent fluid. This fluid, however, soon becomes turbid thus converting the vesicle into a pustule over which the raised epidermis stretches as a thin, glistening membrane. In the degree that the fluid in the pustule increases in amount the pustule will increase in size both as regards its diameter at the base and its elevation. In places protected from mechanical insults such pustules may become hemispherical in shape and have a base two or three millimeters or more across. In some cases the lines of scarification may develop into a marked cord-like swelling throughout their course reaching a width of as much as one-half inch at the base and exhibiting a deep livid red color before pustules may appear thereon. In other cases the swelling is rather diffuse and may reach a thickness of one-half inch. The pustules develop, not only along the line of scarification, but also appear adjacent thereto or indeed some distance removed therefrom. The location of the development of the primary pustules thus shows that while the entrance of the virus has undoubtedly been aided by a slight rupture in continuity of the epidermis in the form of a scarification, yet the development of secondary pustules extends beyond such portal of entry to the apparently unbroken skin. It is beyond the scope of this publication to discuss the problem whether the virus traveled to the location of the new lesions through the tissue or whether it has found a portal of entry through the apparently unbroken epidermis, but our observations indicate that its entry through the apparently unbroken epidermis is entirely possible. The gross appearance and the manner of development of the lesions to this stage resemble those observed in the vaccina-variola group of disease.

The pustules do not all develop simultaneously but new vesicles and pustules may appear from day to day for a week or longer so that all stages of development may be seen side by side. During this time not only the tract of the scarification may become closely studded with pustules but they may also develop some distance removed therefrom so that in the end we may have a broad band of irregularly placed pustules of

which the original scarification approximately forms the middle line. But long before the last pustules have developed, those formed first will have ruptured either spontaneously or from mechancial interference and have scabbed over. It is most likey that the virus escaping from such ruptured pustules is to a greater or less degree spread to adjoining parts of the skin and contributes largely to the development of additional pustules. Thus, it has been repeatedly observed by us that if the original scarification and inoculation was made on the under surface of the tail in goats after the first pustules had ruptured at the original point of inoculation, new pustules would soon thereafter appear on the skin below the anus or vulva at a point far removed from the original site of inoculation but nevertheless subject to being soiled with the escaping content of the pustules.

When the thin glistening layer of epidermis over the pustules breaks and the contents escape, a lesion is formed in which the more vital layer of the skin is exposed and is now subject to secondary infection, particularly by pus organisms which are found everywhere in great abundance. As a rule, however, the content of the ruptured pustules, so far as it is not mechanically removed, and the cell debris, augmented by additional purulent material oozing from the exposed dermis, gradually dry up and form a scab. Naturally the injury set by the virus which led to the formation of the pustules is not repaired with the rupture of the latter, but rather the reaction in the skin will continue until normal conditions are again restored. During the duration of this reaction we will find that the natural protective forces of the body, the polynuclear leucocytes and kindred cells in addition to excessive amounts of serum have been concentrated at the site of attack of the virus and now force their way to the surface acting as scavengers of the polluted area. As they continue to arrive at the surface of the still viable tissue and beneath the already formed scab, they in turn will also become dry and continue to add to the increasing thickness of the latter. In addition to these changes the Malphigan layer of the skin will begin to proliferate, thus forming excessively heavy layers of epithelia which dry out and also add continually to the thickness of the scab; the dermis itself becomes thickened, hypertrophic, and thus raised several millimeters above the general level of the surrounding skin. This becomes particularly apparent when one forcibly removes some of the dry scabs, for one will find under them a raw, bleeding surface markedly elevated over the adjoining healthy skin. The development of the lesions thus have, in the acute stage, all the characteristics of a suppurating dermatitis. At times pus may be found to ooze or may be expressed from under the scabs.

In many instances the development of individual papules can not be observed along the line of scarification. In such cases the line of scarification as a whole or parts thereof shows, on the third or fourth day, increased redness and slight swelling. Both of these manifestations increase in intensity from day to day so that the scarifications soon stand out prominently as deeply bluish-red ridges along which no indication of pustule formation or suppuration can be detected. As these reddened

prominent ridges increase in thickness they also increase in width, sometimes attaining a width of one-half inch. At the end of a week or ten days the surface of these ridges begins to get dry and hard, thus ushering in the scab formation.

Scab formation as just detailed is often rather prolific. The scabs or crusts may reach a thickness of one-half inch or more remaining confined, of course, to areas near the site of infection or to which the virus spread by contact with the contents from the ruptured pustules. They are dry, hard, brown to dark-brown, tightly adherent to the underlying tissue and when forcibly removed leave a raw, elevated, bleeding surface. As time goes on they become cracked and fissured on the surface, from which they gradually crumble away until, at the end of three or four weeks or more, they have entirely disappeared without leaving a scar.

Fig. 1. Scab formation on lips in kid following artificial transmission.

The lesions following artificial transmission as described are those observed in a fairly marked reaction. In many instances, however, much milder lesions will be observed following such inoculation and these vary somewhat in their appearance from animal to animal. In many instances only papules that did not progress into the vesicle or pustule stage were observed. These papules appear in the skin on the inoculated area as hard knots two to three millimeters in diameter, project hemispherically above the surface of the surrounding skin, and later become covered with a thin, dry, brownish scab. This thin scab finally drops off and the knots gradually disappear in the course of the next few weeks. In another type of lesion we have observed a reddening of the skin at the point of inoculation and more or less pustule-formation, but the scab forming over such pustules remained very thin and had a tendency later to peel off as a whole instead of becoming crumbly, as happens in cases where the scab reaches an appreciable thickness. In a third type of light lesion observed the scabs forming after the appearance of pustules would become one or more millimeters thick, would always be dry with no indication of pus-formation under them, remain gray in color, adhere tightly to the somewhat thickened skin, and have a scaly appearance. These scales gradually drop away from the surface as healing progresses.

Lesions on the mucous membrane of the mouth have been less frequently observed by us. In the mouth the ulcerous lesions developing at the point of infection are constantly kept moist so that the exudate from them and the proliferating epithelial cells have no opportunity to become

desiccated but instead remain soft and moist and are constantly being more or less completely removed by the necessary movements of the tongue and other mouth parts against one another while the animal is eating or drinking. The lesions, therefore, show no scab formation but on a reddened mucous membrane there appears a somewhat elevated, creamy, whitish-yellow, spongy, membranous deposit. The tissue underlying such deposits bleeds very readily when it is touched. These lesions can be produced on the mucosa by inoculation similarly as on the skin.

It is significant that in all our transmission experiments where the site of inoculation was either the internal aspect of the thigh or the under surface of the tail, the lesions remained localized to such point of inoculation. In no case did lesions develop on the lips.

TRANSMISSION OF THE DISEASE

Experiments to determine the nature of the disease were first undertaken by us in September, 1928. It could readily be shown that the disease was infectious. The first attempt to transmit the disease by scarification of the lips of lambs and by applying emulsions made from freshly collected scabs were successful in reproducing the disease. Such artificial transmission of the disease has since been repeated on numerous occasions. In one instance soremouth was also produced by scarifying the lips of a lamb by pricks with a cactus thorn and vigorously rubbing into it dirt from the floor of a sheep stall in which soremouth cases had been kept. From this, one might infer that the virus might also be found in pastures where the infective material has dropped from the animal and there constitutes a source of infection for other animals as they graze over such areas. How long the virus may remain alive in the soil and litter of sheep sheds has not been established. It is known that outbreaks of soremouth do not appear on the same premises year after year but sometimes a year or more may intervene between outbreaks. Whether in such cases the virus had died out and was later re-introduced from an outside source is not known. However the fact that the disease does not appear in any one year on infected premises does not mean that such premises are permanently free from the disease.

In our experiments, as will be seen from Table 1, the area to be inoculated was scarified in every instance. That lesions can also develop on unscarified areas has already been pointed out. In view of the latter observation it must be considered doubtful whether sheep or goats in order to become infected must first injure their lips on coarse feed or otherwise before infection can take place. That such injury to the lips might aid infection in establishing itself is quite plausible.

Our attempts to transmit the disease to guinea pigs have been unsuccessful. That other investigators have failed to transmit the disease to other animals has already been recorded. It may be pointed out in this connection that the disease has never been observed in cattle, although they have frequently come in close contact with infected sheep or goats. References are contained in the literature (3, 4) to the effect that lesions

resembling soremouth have been observed in persons coming in close contact with the disease in sheep. We have observed two similar instances, but as yet have no definite proof that such lesions were due to soremouth virus affecting sheep or goats.

Table 1. Experimental transmission of soremouth; point of inoculation scarified.

Date	Number of animal	Source of Virus	Site of inoculation	Result
9/15/28	Lamb 17	Dirt from sheep shed	Lips	Soremouth
	Lamb 18	Scabs	Lips	Soremouth; raw, bleeding ulcers and false membranes also on mucosa of lips.
	Lamb 19	Scabs		Soremouth; raw, bleeding ulcers and false membranes also on mucosa of lips
10/ 1/28	Lamb 26	Scabs from No. 17	Lips	Negative
	Lamb 27	Scabs from No. 18	Lips	Negative
	Lamb 28	Scabs from No. 19	Lips	Negative
11/22/28	Lambs 16, 17, 18, 19, 20, 30, 31	Dirt from sheep shed	Lips	Negative
12/24/28	Lambs 67, 68, 69	Scabs	Lips	Soremouth
1/ 3/29	Kid 76 Lambs 77, 79, 80	Virus No. 68	Lips	Soremouth
3/ 2/29	Ewe 294	Scabs	Lips	Mild soremouth
	Ewe 295	Scabs	Lips	Severe soremouth
5/ 2/29	Kid 969	Virus No. 967	Lips	Soremouth
	Nanny 970	Virus No. 967	Lips	Soremouth
	Nanny 977	Not inoculated, nursing No. 969		Developed soremouth lesions on teats
5/ 2/29	Guinea pig 971	Virus No. 967	Lips	Negative
5/16/29	Guinea pig 1030	Virus No. 969	Lips	Negative
	Guinea pig 1031	Virus No. 969	Lips	Negative
	Guinea pig 1032	Virus No. 969	Lips	Negative
5/20/29	Kid 1057	Virus No. 969	Lips	Soremouth
	Nanny 1058	Not inoculated, nursing No. 1057	Lips	Developed soremouth lesions on teats
4/25/29	Goat 1	Sheep	Lips	Light scab formation
	Goat 2	Sheep	Inguinal region	Marked scab formation
5/22/29	Goat 3	Goat	Inguinal region	Marked scab formation
6/15/29	Kid 4	Unknown	Inguinal region	Marked scab formation
	Kid 5	Unknown	Under tail	Doubtful
	Kid 6	Unknown	Under tail	Light scab formation
	Kid 7	Unknown	Under tail	Doubtful
9/13/29	Kid 4 (retest)	Unknown	Inguinal region	Marked scab formation
	Kid 8	Unknown	Inguinal region	Marked scab formation

DIFFERENT STRAINS OF VIRUS

The question whether one strain of virus must be recognized for the sheep and one for the goat has not yet been decided. We were able to transmit the sheep virus obtained from natural cases of soremouth in the sheep to goats and vice versa but believe to have observed a much milder development of the disease in the sheep when goat virus was used and

vice versa. Table 2 shows the reactions obtained in sheep when inoculated with fresh goat virus.

Table 2. Susceptibility of sheep to goat virus. Sheep inoculated Nov. 20, 1931 with fresh goat virus in inguinal region after scarification.

Animal number	Reaction 11/25	11/27	11/29	12/4
1	Doubtful	Many pustules	Pustules and scabs	Pustules dried up but no brown scabs formed
2	Red	Doubtful	Doubtful	Doubtful
3	Pustules	11 pustules	Meager scab formation	Scabs dropping off
4	Red	Pustules	Meager scab formation	Scabs dropping off
462	Pustules	3 pustules	Meager scab formation	Scabs dropping off
463	Red	Pustules and scabs	Meager scab formation	Scabs dropping off

Although numerous pustules were formed in two of the six sheep inoculated, yet in the animal showing the most numerous pustules they dried up without leaving a dry, brown scab; and in the animal showing the next largest number of pustules the thin scabs formed were already dropping off nine days after the appearance of the pustules. When such a marked pustule formation took place in the goats from which the virus used to inoculate these sheep was taken a very heavy scab formation always resulted, which in all probability would not have cleaned up in less than three or four weeks. Field observations by us also indicate the existance of two different strains of virus and a similar observation is recorded in the literature (4), although not so interpreted. Jacotot (1), although he does not cite any evidence, is of the opinion that sheep appear more resistant to infection than goats. We expect to continue our researches regarding this point.

SUSCEPTIBILITY OF ANIMALS

On the range where the disease is endemic, outbreaks in flocks are usually limited to the young animals up to a year old. Of these it is estimated that as many as 90% to 95% may develop more or less marked readily recognizable lesions on the lips. In our experiments with young goats we observed a marked variability in the "take" of the disease which could not be explained on the basis of insufficient exposure or a variability in the virulence of the virus. We believe that our technic of inoculation, consisting of a scarification of the skin and vigorously applying an emulsion of relatively fresh triturated soremouth scabs, subjects the animal to as severe an exposure to the infection as it may encounter in the field, and yet we were not able to reproduce the disease in every case. We always inoculated at least two animals at the same time with the same virus and sometimes found only one of the two

to develop the disease. Moreover we found animals that failed to develop the disease even after a number of inoculations. As an example we cite kids Nos. 413, 431, and 450 shown in Table 3. One of these kids, No. 431, resisted infection in three inoculations, and two, Nos. 413 and 450, in two inoculations; while all other animals (five or more) in the same experiment developed the disease. This finding suggests that some animals may possess a natural immunity and would explain why not all young animals in a flock contract the disease.

Table 3. Showing resistance of some animals to (artificial) inoculation.

Kid No.	Date inoculated, 1931.		
	8—3	10—1	11—20
413	—	—	11 papules about 2 mm. in diameter
431	—	—	—
450		—	—
Controls	+	+	+

It is only seldom that gross lesions are seen in older animals on the range but on several occasions such were found upon closer examination of apparently clean animals. Information is not available whether these animals had a previous attack of the disease or whether they had been exposed and escaped infection or had never come in contact with the disease. In order to secure some information on the susceptibility of aged animals, experiments were undertaken on aged goats outside of the endemic area on animals that had been raised on the experimental grounds and were known never to have been exposed to the virus. The results of these tests are shown in Tables 4 and 5. Of the 26 animals inoculated 12 showed a marked positive reaction, 10 a doubtful to mild reaction, and 4 showed no reaction. Since all of these animals were inoculated with freshly collected virus the results clearly indicate that there is a marked variation in the susceptibility of older animals.

Table 4. Susceptibility of aged goats to soremouth. Inoculated Oct. 1, 1931 with goat soremouth virus by scarification of skin on under surface of tail.

No. of animal	Dates of examination (1931) and results.						
	10/5	10/7	10/8	10/9	10/10	10/17	10/24
418	—	—	—	—	—	—	—
434	—	—	—	—	—	—	—
452	—	—	—	+ ?	3 papules	3 papules	Clean
453	—	+	+	++	++	++	Clean
355	+ ?	+	++	++	++	++	Clean
455	—	—	+	++	++	++	Clean 10-30-31

FILTRATION EXPERIMENTS

After we had clearly established the infectiousness of soremouth we began to cast about for the probable cause. Our observations of the nature and development of the lesions let it appear likely that the cause of the disease is not an ordinary bacillus and would likely be very difficult to isolate. Indeed the literature appearing in the meantime sub-

Table 5. Susceptibility of aged goats to sooremouth. Inoculated Oct. 31, 1931 with goat soremouth virus by scarification of skin on under surface of tail.

No. of animal	Tooth age	Date of examination (1931) and results								
		11/2	11/5	11/7	11/9	11/11	11/16	11/18	11/20	11/25
406	8 short	—	—	—	—	—	—	—R		
410	8	—	+	++	++	++	++	++	++	Cleaning
414	8	+?	—	—	+?	+?	—	—R		
421	Broken mouth	+?	+?	+	+	++	++	++	Cleaning	Clean-Released
432	8	—	—	+?	+	—	—	—R		
424	8	—	—	+	—	—	—	—R		
426	8	—	—	+?	+?	+	—	—R		
428	8 short	+?	+?	+	++	++	++	++	Cleaning	Clean-Released
430	8	—	+	+	+	+	—	—R		
432	8	—	—	—	+?	+	—	—R		
436	8	—	—	—	—	—	—	—R		
438	8	+?	—	—	+?	—	—	—R		
440	8	—	—	+	++	++	++	++	Cleaning	Clean-Released
442	6	—	—	+	+?	—	—	—R		
444	Broken mouth	—	—	—	+?	+?	—	—R		
457	8	—	++	++	++	++	++	Cleaning	Clean	Released
458	8	—	+	++	++	++	++	++	Clean	Released
459	8	—	—	+	++	—	—	—R		
460	8	+	+	++	+?	++	++	++	Clean	Released
461	8	—	+	++	++	++	++	Cleaning	Clean	Released

R=Released from experiment; —=Negative; +?=Doubtful; +=Mild lesions; ++=well-marked lesions.

stantiated our belief and contained reports of the filterability of the virus. In order to more definitely establish the identity of soremouth as observed by us with the disease described as contagious ecthyma, etc., filtration experiments were undertaken. Six experiments were run, three using the Mandler diatomaceous filter and three using the Seitz asbestos filter. In each case scabs recently collected and preserved dry in the dark were finely ground in a sterile mortar. Physiological salt solution was then added and the grinding continued about 5 minutes longer. This emulsion was then diluted with physiological salt solution to give a dilution of about one to fifty on the basis of dry scabs used. One portion of the emulsion was then passed through a Seitz filter and another portion through a Mandler filter. Filtration was started within an hour after the emulsion had been prepared. In each case the unfiltered residue of the Mandler filter was used to inoculate the control animals. Filtration through the Seitz filter was accomplished under pressure and required only a few minutes. Filtration through the Mandler filter was by suction and required about thirty minutes. Only enough material was passed through the respective filters to suffice for the inoculation of three animals from each. Inoculation of the animals was undertaken within an hour after the completion of the filtration with the exception of Experiment 1, in which case the filtrates together with the residue for the inoculation of the control animals was kept in a refrigerator for 24 hours. Three animals were inoculated on the under surface of the tail after scarification from each of the two filtrates, while 4 animals were inoculated in a similar manner with the residue. The results are recorded in Table 6.

Table 6. Filtration experiments.

Animal number	Date 1931	Mandler Filter	Seitz Filter	Controls
First experiment				
419	6-12	—		
431	"	—		
439	"	—		
413	"		—	
427	"		—	
433	"		—	
420	"			+
435	"			+
441	"			+
451	"			++
Second experiment*				
407	7-3			++
437	"			++
446	"			++
447	"			+++
Third experiment*				
423	7-21			+++
425	"			+++
435	"			+++
445	"			+++

*Only control animals are listed in 2nd and 3rd experiment. To test filterability of virus the same animals were used as in Experiment No. 1.

Since none of the animals inoculated from the filtrate developed lesions of soremouth, the same animals were inoculated from the same kind of

filtrate in the next succeeding filtration experiment while 4 new controls were used. After the completion of these experiments, the animals inoculated with the filtrates were inoculated with unfiltered virus to test their susceptibility to soremouth. In each instance only two of the three animals used for each of the two kinds of filtrate developed well-marked lesions while the third animal in each group, already referred to in our previous discussion as No. 413 from the Seitz filtrate and No. 431 from the Mandler filtrate, failed to develop lesions. Since these two animals resisted two later inoculations with known potent virus, the possibility arises that they might have contracted an immunity without the development of grossly recognizable lesions. The results of the other two animals in each experiment would indicate, however, that this is not the case. We expect to investigate this point further.

The results of our filtration experiments were thus all negative. In view of the results from similar filtration experiments recently recorded in the literature this outcome was not surprising and can only be interpreted as emphasizing the findings of others that the virus is at least very difficult to filter. Additional experiments will be undertaken to determine whether our results can be brought into complete agreement with those of others.

IMMUNITY TESTS

The question of immunity in soremouth has interested us from the time we were able to show that the disease is infectious. The fact that from a practical standpoint only young animals contract the disease on the range and that the mother animals usually remain free from lesions, although they are in close association with their nursing young, already suggests that an immunity develops. Since the disease itself is one of the skin, any immunity established is likely an immunity of that organ, which would develop regardless of the point of infection. With this possibility in mind we sought to transfer the development of the lesion from the lips to a point where it would not interfere with nursing or prehension of food and water. The most desirable place to set the lesion is, of course, one devoid of hair or wool, such as the under surface of the tail or the internal aspect of the thigh. For the purpose of establishing an immunity, either would be satisfactory.

Our first experiments to test the practicability of establishing such an immunity were run in 1929. The outcome of these were not very satisfactory. Further work on this point was postponed until 1931, when more extensive tests were undertaken. The results of these tests are recorded in Table 7.

The duration of the immunity following artificial inoculation has not been followed in our animals over a very long time largely because it was impractical for us to hold them over an extended period for such tests. That immunity does not always follow the first inoculation even though fairly marked lesions are established is shown by animal 4, Table 1, and animals 407 and 451, Table 7. Animal 4 was checked only

once three months after the original inoculation while animals 407 and 451 were checked twice, the first time 41 days and 45 days respectively after the original inoculation. All three animals again showed well developed lesions upon the first check but when animals 407 and 451 were again checked 89 and 58 days after the first immunity test they did not again react.

Table 7. *Immunity test for soremouth on goats; inoculated on under surface of tail after scarification.*

Kid No.	Date of first inoculation, 1931	Inoculation for immunity test: Date, 1931	Inoculation for immunity test: Reaction
407	5—22	7— 3	+
407†	7— 3	10— 1	—
411	6— 2	"	—
419	8— 3	"	—
423	7—21	"	—
425	7—21	"	—
427	8— 3	"	—
433	8— 3	"	—
435	7—21	"	—
437	7— 3	"	—
439	8— 3	"	—
445	7—21	"	—
446	7— 3	"	—
447	7— 3	"	—
451	6—19	8— 3	+
451†	8— 3	10— 1	—
Controls for final test			
413*		10— 1	—
415		"	+
417		"	+
420		"	+
429		"	+
431*		"	—
441		"	+
448		"	+
450*		"	—

*Regarding reaction of these three animals see also Table 3.
†These are second trial with the same animals.

Encouraged by these results we decided to undertake a field test. The ranch selected was one on which both sheep and goats were kept and on which soremouth had occurred among the goats but not among the sheep during the previous year. Two hundred lambs and 77 kids were inoculated on the under surface of the tail with goat soremouth virus and conspicuously marked for proper identification; three hundred lambs and 70 kids were left for controls. The lesions developing among the inoculated animals were not checked for severity. Some three weeks later the uninoculated control kids began to show soremouth lesions on the lips, and by the 26th day it was estimated that 80% of the control kids showed such lesions, some of them so severe that the owner found it necessary to treat them. No soremouth occurred among the lambs left as controls. The owner of these animals was very much encouraged by the outcome of the test and as the result of it we have inoculated a large number of lambs during the present season with the view of restricting the lesions to a part of the body where they do not interfere with the taking of nourishment and where they can be set early

enough in the season before the screw-worm fly becomes active. In this way we expect to greatly reduce the losses from soremouth. As this manuscript goes to press, results of these vaccination experiments also indicate that vaccination is highly successful.

Much work remains to be done on the degree of immunity developed following artificial inoculation, especially on the degree of the visible gross reaction necessary to establish an immunity that will stand up in the field. We have especially in mind to determine whether the mild types of lesions described as sometimes following artificial inoculation will establish a satisfactory immunity or not. This whole question is, of course, closely bound up with the virulence of the virus, the production of a suitable virus properly attenuated for use in the field, the susceptibility of the animal, and cross immunity between strains of virus developing on sheep and goats.

TREATMENT

Medical treatment for soremouth at its best is unsatisfactory. This is to be expected from the nature of the disease. The destruction of the virus would be the logical thing to strive for, but by the time lesions are observed the greater part of the virus is already located within the skin out of reach of any medical treatment applied to the surface. If the owner wishes to apply a germicide at this time, one that has deeply penetrating powers should be selected such as tincture of iodine or an alcoholic solution of gentian violet. The germicidal power of pine tar may also be utilized and is best diluted with some oil. For this purpose used crankcase oil may be taken in the proportion of one part pine tar and two parts oil. This preparation may also be used later when scab formation has already set in. It has a tendency to soften the scabs and thus keep the lips more pliable. A better preparation for this purpose is a mixture of one part of tincture of iodine to two parts of glycerine. Other bland fatty or oily substances to which some non-irritating antiseptic has been added serve the same purpose. Repeated applications of the selected treatment are indicated.

SUMMARY

Soremouth (Contagious Ecthyma) of sheep and goats is very prevalent in the sheep- and goat-raising area in western Texas, and is also found in other states of the Union as well as over large areas in other sheep-raising countries. The disease can readily be transmitted by emulsions made from the dry scabs formed as a result of the infection when applied to the scarified skin. The course of the disease with its attendant lesions is described.

The disease is most prevalent among young animals up to one year of age but a large percentage of much older animals can be readily infected artificially. The mortality from the disease is very low but

indirect losses resulting from screw-worm infestation, stunting of young animals, and feed-lot shrinkage may be very high.

The nature of the virus has not yet been determined. Some investigators report that they succeeded with difficulty in filtering the virus through various bacterial filters. We failed in three attempts to pass the virus through Mandler and through Seitz asbestos filters. The disease can be transmitted from sheep to goats and from goats to sheep but the goat virus does not appear to be as virulent for the sheep as sheep virus and vice versa. This points to the existence of a sheep virus and a goat virus.

In our experiments recovery from the disease left a high degree of immunity, the maximum duration of which is still unknown. Whether a cross immunity exists between the possible sheep and goat strains of virus has not yet been determined. Although on the range the lips are the principal site of the lesions, nevertheless they occasionally also appear on other parts of the skin and can be produced artificially on any part of the skin, where they will remain localized. We are taking advantage of this fact and expect to test out the practicability of immunizing the young animal on some part of the body other than the lips before screw-worm time arrives in the spring.

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